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Analysis of Asynchronous Wall Motion by Regional Pressure-Length Loops in Patients With Coronary Artery Disease

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The progression of regional dysfunction during angina pectoris was studied in eight patients with coronary artery disease. Single plane left ventriculograms were obtained using a high fidelity micromanometer-tipped catheter both at rest and immediately after rapid cardiac pacing. Each image of the left ventriculogram was digitized and transferred to a computer. The boundary of the ventricular cavity was automatically determined and sequentially superimposed. Regional shortening was quantified by a radial coordinate system originating at the center of gravity of the end-diastolic silhouette. Thirty-two radial grids were drawn around the center of gravity, and the length of each radial grid was measured to characterize the centripetal motion of a given surface point. Each radial length was then plotted simultaneously and continuously against left ventricular pressure to generate a pressure-length loop.

The area of the pressure-length loop provided an index of regional myocardial work. In the ischemic ventricle, the loops exhibited a striking deformity in configuration. Prolonged relaxation of ischemic segments was associated with outward motion of the normal segments. Shortening of the normal segment occurred earlier than that of the ischemic segment associated with its stretch. Thus, the loops of the two areas inclined in opposite directions. Pacing stress increased the magnitude of hypofunction in the potentially ischemic area, the average extent of shortening being reduced by 30% and the segmental work by 25% ($p < 0.005$). In the normal area, contrary to the significant increase in segmental shortening (20% above control values [$p < 0.005$]), the average segmental work remained at 7% below control values because of an augmented deformation of the loop. The isovolumic asynchrony of the left ventricle due to reciprocal contraction and relaxation in opposing myocardial regions profoundly modifies the shape of the regional pressure-length loops and external segmental work.

Contractile function of the ischemic ventricle has been defined by the dynamic shortening and lengthening characteristics of the regional myocardial segment in both experimental and clinical settings (1–3). However, the mechanical property of cardiac muscle has been traditionally evaluated in two ways by measuring: 1) the shortening of the muscle fiber, and 2) the development of tension within the muscle (4,5). When the heart as a whole is compared with the isolated muscle, cardiac dimension (volume or segment length) and intracavitary pressure are analogous to muscle length and tension, respectively. In the ejecting ventricle, the stroke volume (or extent of wall shortening) is analogous to the extent of shortening of the isolated muscle. The instantaneous left ventricular pressure can be plotted against the corresponding left ventricular volume throughout the cardiac cycle. The resulting pressure-volume loop provides a convenient framework for understanding global contractile functions of the myocardium (6–8). The integral pressure with respect to cardiac dimension during ejection allows for an assessment of the total ventricular work. However, in the presence of coronary artery disease, dysfunction of the ischemic myocardium always accompanies compensatory augmentation of the contractile function of the normally perfused myocardium (2,3), which causes little change in total cardiac function.

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In the present study, we constructed pressure-length loops of the regional myocardium with the use of left ventriculography by plotting instantaneous pressure against radial length to evaluate the regional nature of the contraction abnormalities in patients with coronary artery disease. Special attention was directed to analyze the modification of segmental pressure-length loops by rapid cardiac pacing in the myocardium potentially subjected to ischemia during a stressed state.

Methods

Study patients. The study was performed in eight patients with coronary artery disease and angina on effort. Patients with global ischemia due to multivessel disease were excluded. All patients were in normal sinus rhythm and all medications were withheld 12 hours before the procedure. Informed written consent was obtained from each patient and there were no complications as a result of the study.

Catheterization procedure. Cardiac catheterization was performed using the left brachial approach with the patient in a fasting state and after premedication with 5 mg of oral diazepam. After conventional diagnostic right and left heart catheterization, coronary arteriography was performed using the Sones technique. A pacing catheter was positioned in the right atrium. A high fidelity micromanometer-tipped catheter (Micro-Tip, Millar Instruments) was then introduced into the left ventricle through the left brachial artery, which allowed for simultaneous high fidelity pressure measurement during angiography. After confirmation that ventricular pressure had returned to baseline level after coronary arteriography, left ventricular cineangiography was performed in the 30° right anterior oblique projection, using a Philips 9 inch (22.86 cm) image intensification system. Left ventricular opacification was achieved by injecting 25 to 40 ml of radiopaque contrast medium (80% Angioconray) through a Millar angiographic catheter at a rate of 12 ml/s. Films were exposed at a rate of 60 frames/s with an Ari 35 mm cine camera while the patient was gently holding breath. During each cineangiographic study, high fidelity left ventricular pressure, electrocardiogram, cineangiographic frame markers and an injection marker were simultaneously recorded. Two lead markers were placed on the image intensifier as fixed references for superimposition of the images. An adequate recovery time was allowed for left ventricular pressure to return to baseline level. Atrial pacing was initiated at a rate of 90 beats/min and was increased in steps of 30 beats/min every 2 minutes. In patients in whom atrioventricular block developed during atrial pacing, right ventricular pacing was substituted. Pacing was stopped with the occurrence of chest pain. If well tolerated, pacing was continued at a rate of 150 beats/min for 6 minutes. The second angiogram was obtained immediately after the cessation of pacing in exactly the same manner as in the control state (3).

In one patient, angiography was repeated after the administration of sublingual nitroglycerin and demonstrated a remarkable improvement of the initially observed abnormal wall motion. Data are shown in figures only as a reference of normally synchronized contraction.

Analysis of Data

Boundary detection. The method of automatic processing of cineangiograms has been described elsewhere (3,9,10). Briefly, the left ventricular images on cine film were transferred to a computer through a flying spot scanner and were stored on a magnetic disc. Each digitized image consisted of 128 × 128 pixels with gray levels of 256 values. A gradient image was then obtained by spatial differentiation of gray levels. Assuming that the abrupt change of the gray levels occurred at the boundary of the ventricular silhouette, the points with the maximal gradient value were traced to delineate the ventricular boundary.

For the algorithm for each computer tracing of edge points, two weight coefficients were introduced to multiply the corresponding derivative value. One is denoted as the directional weight coefficient, which enables avoidance of an abrupt change in direction of the edge tracing. The other is the coefficient to define the depth of search in which information of several remote points is to be referred. The additional global guidance was obtained from the preceding frame. All these procedures closely imitate visual detection of a boundary by the human eye (3).

Analysis of global left ventricular function. Left ventricular volume (V) was calculated by a modification of Kennedy's formula (11):

$$V = 0.687 \times C^3 \times A^2/L + 1.9 \text{ ml},$$

where A is the area of the ventricle calculated from the amount of pixels surrounded by the left ventricular boundary, L is the longest measured length between the midpoint of the aortic valve and the apex and C is the linear correction factor for the magnification of a unit of length (1 pixel), which was derived from a comparison with the known area of the filmed 1 cm^2 grid placed parallel to the tube at the position of the heart.

The calculated volume of each frame was synchronized to corresponding pressure by a simultaneously recorded exposure marker throughout the cardiac cycle to obtain the pressure-volume loop (Fig. 1). The left ventricular stroke work was calculated by integrating the area of the pressure-volume curves.

Analysis of regional left ventricular function. Sequential ventricular silhouettes were superimposed on the end-diastolic frame throughout the cardiac cycle by using two external reference markers. Significant movements of the heart can be a cause of error in interpretation, and an

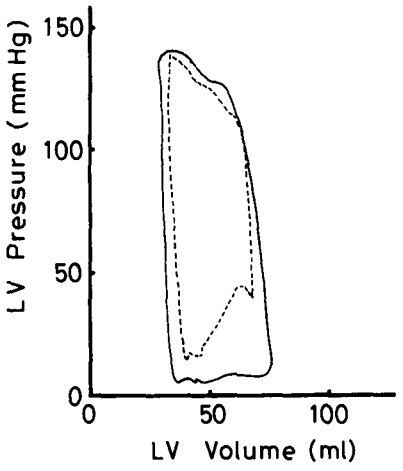

Figure 1. Pressure-volume loops obtained before (**solid line**) and during (**dotted line**) angina in a patient with obstruction of the left anterior descending coronary artery. Despite development of regional wall motion abnormalities with pacing stress, the pressure-volume loop maintained a normal configuration with a counterclockwise rotation. There is a striking elevation of the diastolic pressure-volume curve with resultant reduction of the area enclosed by the loop. LV = left ventricular.

area of asynergy might rotate in and out of the perimeter seen in profile. However, these problems are common to all acceptable methods of wall motion analysis by cineangiography, and movement of the heart within the chest has been shown to be minimized when respiration is suspended during filming (12). In each superimposed ventricular image, 128 radial grids were drawn from the center of gravity of the end-diastolic silhouette to the endocardial margin. Measurement of the length of each grid line throughout the cardiac cycles made it possible to analyze the contraction and relaxation of specific segments of the left ventricular myocardium (3) (Fig. 2).

The length of each radial grid plotted against time was handled in the same way as an overall volume-time curve to construct the pressure-length loop (Fig. 3 and 4). The area inscribed in this loop was taken as an index of regional myocardial work in units of mm · mm Hg (13–17). Thus, a pressure-length loop was originally generated for 128 radial grids, but was reduced to every fourth loop, resulting in a display of 32 loops over the entire ventricular circumference (Fig. 3). Excluding the area of aortic and mitral valves, the outline of the left ventricular cavity was separated into five sections that roughly corresponded to five segments (anterobasal, anterolateral, apical, diaphragmatic and posterobasal) defined in a reporting system described by the American Heart Association (18). The stroke excursion and the segmental loop area were averaged in each of the five sections that included four or five loops. To analyze the effect of pacing stress on the ischemic myocardium, the potentially ischemic section corresponding to the known coronary lesions, in which active shortening was preserved at rest, was selected, and the response to cardiac pacing was compared with that of the normal section perfused with intact coronary arteries. In the patients with overt myocardial infarction, the section that included the central ischemic region was excluded, because neither dyskinetic nor akinetic motion of the definite infarction area was modified substantially by pacing stress.

All data were expressed as mean ± standard deviation. Statistical comparisons were made by a paired *t* test.

Results

Data obtained in all eight patients are listed in Table 1. In all patients, typical anginal pain developed during pacing tachycardia. In an area perfused with a diseased vessel and potentially subjected to ischemia during a stressed state,

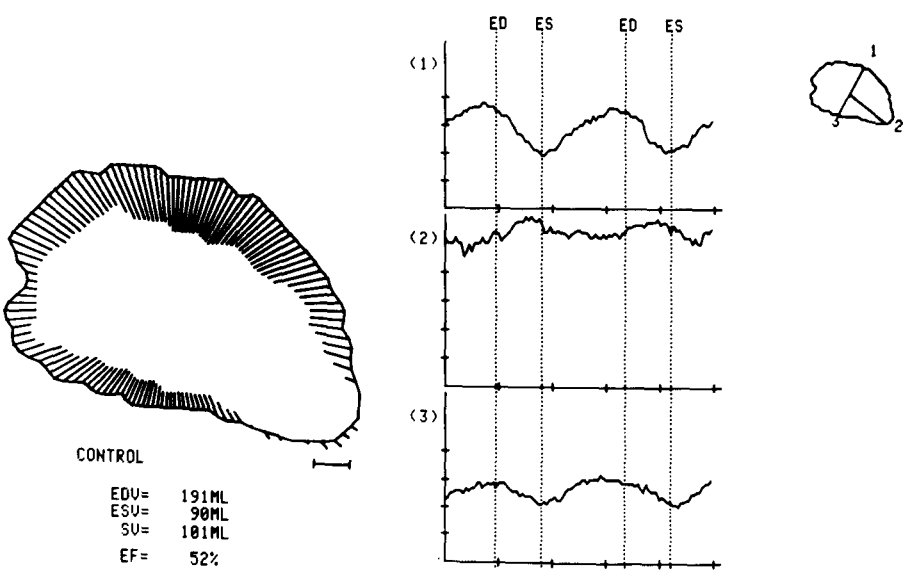

Figure 2. Superimposed end-diastolic and end-systolic boundaries of the left ventricle (**left panel**). Sequential ventricular images are superimposed and 128 radial grids are drawn from the center of gravity of the end-diastolic silhouette to the endocardial margin. The continuous measurement of the length of grid lines enables a quantitative description of the segmental centripetal motion over the entire circumference. The representative time-length plots in the three specific grid lines in the anterior (1), apical (2) and inferior (3) wall are shown on the **right**. The location of each radial grid is located in the **upper right-hand corner**. ED = end-diastole; EDV = end-diastolic volume; EF = ejection fraction; ES = end-systole; ESV = end-systolic volume; SV = stroke volume.

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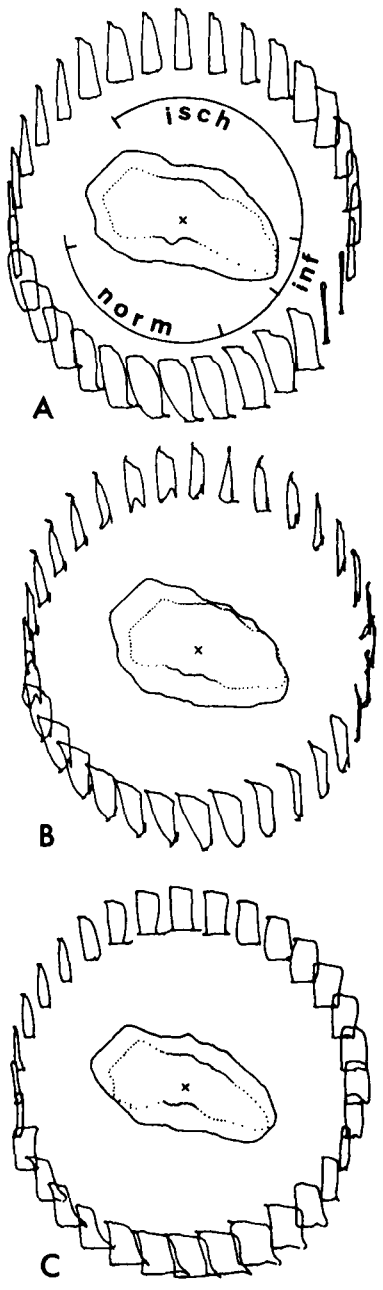

Figure 3. Sets of pressure-length loops constructed by relating each radial length to the phasic pressure as the common variable throughout the cardiac cycle. There were originally 128 radial axes, but these were reduced to every fourth radial axis resulting in 32 loops around the ventricular silhouette. **A,** Data obtained in the resting state. Ischemic (isch), infarcted (inf) and normal (norm) areas are indicated around the perimeter of the ventricle. x = the center of gravity of the end-diastolic silhouette. The apical segment loops in the central ischemic area are considerably deformed. The anterior wall is perfused by well developed collateral circulation from the right coronary artery, and active segment shortening is maintained but coronary reserve is critically limited. Loops in the anterior wall exhibit an inclination toward the right suggesting delayed relaxation due to potential ischemia during the stressed state. The loops in the normally perfused inferior wall represent the inclination toward the left with lengthening of the myocardium during isovolumic relaxation. There is also an ischemic area at the inferior wall between the infarcted and the normal areas but loops are not typical in configuration presumably due to a tethering effect. **B,** Data obtained during an anginal attack induced by rapid cardiac pacing. The deformation of the pressure-length loop is now expanded over the entire anterior wall. The loops in the inferior wall exhibit a more marked inclination toward the left and are associated with augmentation of systolic excursion. **C,** After an adequate time was allowed for recovery from the effect of repeated angiography, nitroglycerin, 0.3 mg, was administered sublingually to this patient. All the loops obtained after nitroglycerin are rectangular in configuration and have a counterclockwise rotation.

cardiac-pacing elicited acute reversible abnormalities in contraction that are usually absent at rest. There was no substantial change in the function of the central ischemic area, while shortening of normally perfused area was augmented.

Global function and pressure-volume diagrams. In post-pacing beats, heart rate did not change significantly (66 ± 12 versus 61 ± 14 beats/min). Left ventricular peak systolic pressure remained unchanged, whereas end-diastolic pressure increased from 11 ± 4 (± standard deviation) to 22 ± 4 mm Hg ($p < 0.001$). Although there were no consistent changes in left ventricular end-diastolic volume, end-systolic volume was consistently augmented from 48 ± 16 to 59 ± 20 ml ($p < 0.05$). Stroke volume was unaltered (52 ± 12 versus 49 ± 14 ml), but ejection fraction was reduced from 53 ± 7 to 46 ± 8% ($p < 0.05$).

The pressure-volume loop in the resting state is characterized by a counterclockwise rotation with a rectangular configuration (Fig. 1). From the end-diastolic point at the lower right corner, the pressure increases rapidly with little change in volume (isovolumic contraction). With the onset of ejection, there is a substantial decrease in volume with little change in pressure. At the end of shortening, pressure decreases isovolumically and then both pressure and volume increase simultaneously along the pressure-volume relation at rest until the loop closes at end-diastole.

After pacing, the general shape of the pressure-volume loop was unaltered (Fig. 1). The stroke work was reduced from 0.70 to 0.63 J, largely because of the increased diastolic pressure rather than a reduction in shortening or systolic pressure.

Segmental function and regional pressure-length loops. There was a significant heterogeneity in regional myocardial function of the ischemic ventricle and its response to pacing stress. In the center of the infarcted area, active shortening was abolished and the pressure-length loop represented clockwise rotation or figure of eight inscription, indicating that the work was performed on rather than by the myocardial segment. These changes were not modified by the pacing stress. In the potentially ischemic area surrounding the infarcted area, rapid cardiac pacing temporarily

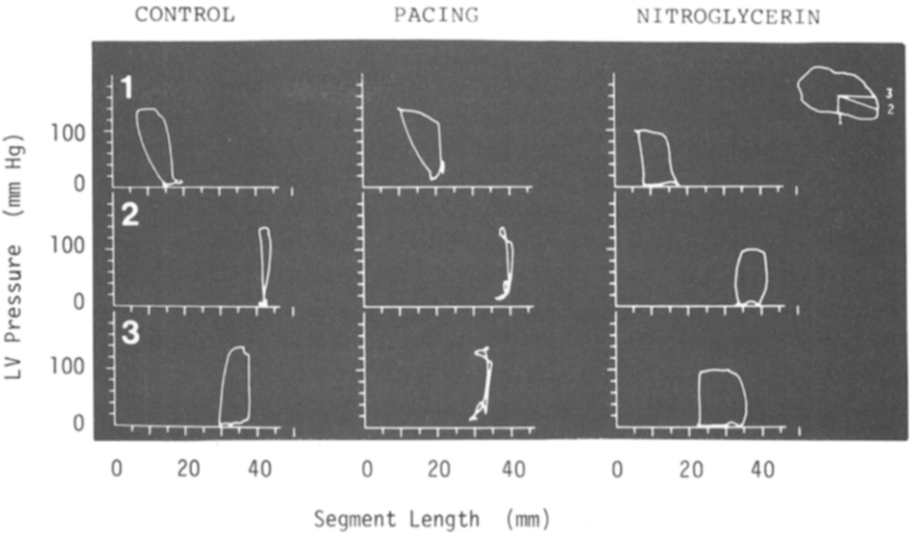

Figure 4. Pressure-length loops in the resting state (control, **left panel**), after rapid cardiac pacing (**middle panel**) and after sublingual nitroglycerin (**right panel**) are shown for three representative segments in the normal (1), central ischemic (2) and potentially ischemic sections (3). The location of each radial grid is indicated in the **upper right-hand corner**. Pacing stress induced striking deformation of the loop in the ischemic segments and inclination of the loop in the normal segment toward the **left**. Thus, change in external work is somewhat diminished. Normal rectangular configuration with counterclockwise rotation was restored in both segments after nitroglycerin. LV = left ventricular.

provoked significant changes. Although the end-diastolic length remained unaltered (32.0 ± 6.8 mm), the end-systolic length was markedly augmented from 26.1 ± 6.6 to 29.8 ± 7.6 mm ($p < 0.01$). Stroke excursion was decreased from 5.9 ± 1.8 to 2.0 ± 1.2 mm ($p < 0.002$)(Fig. 5). The systolic shortening of the potentially ischemic segment was followed by a late additional shortening during the decline of the ventricular pressure. The segment was also lengthened during the isovolumic pressure increase. Accordingly, the pressure-length loop represented an inclination toward the right (Fig. 3 and 4). In post-pacing beats, the sustained shortening during relaxation was exaggerated, and marked deformation of the loop extended over the adjacent area (Fig. 3 and 4). Such alteration of the configuration of the loop was reflected in the segmental work index calculated from the area enclosed by the loop. The mean segmental work index of the potentially ischemic area decreased by 54% from 600 ± 205 to 275 ± 160 mm · mm Hg ($p < 0.001$); this was significantly less than the reduction in the stroke excursion (by 64% from the control state) (Fig. 5).

The loops in the normal area also represented significant deformity. In the resting state, shortening of the normal area began earlier during isovolumic systole and lengthening occurred during isovolumic relaxation. Thus, the pressure-length loop tended to incline toward the left (Fig. 3 and 4). With pacing stress, the end-diastolic length increased from 28.6 ± 6.3 to 30.2 ± 5.4 mm ($p < 0.05$), whereas the end-systolic length remained the same. Stroke excursion was significantly augmented from 8.4 ± 3.7 to 9.8 ± 4.2 mm ($p < 0.005$). Despite this increased shortening, the area surrounded by the loop was decreased from 824 ± 391 to 751 ± 434 mm · mm Hg ($p < 0.005$) (Fig. 5). These changes are largely due to the more exaggerated inclination of the loop, as well as to an elevated diastolic pressure.

Discussion

Regional pressure-length loops in the ischemic ventricle. In the present study, a regional pressure-length loop was generated in the clinical setting by relating segmental shortening of several portions of the left ventricular wall quantified by a radial coordinate system to the corresponding left ventricular pressure simultaneously recorded during angiography. In the normal synchronous contraction as shown in the beat after the administration of nitroglycerin (Fig. 3C), all loops rotated counterclockwise during ventricular systole. Loops were rectangular in formation with minimal change in dimension during the isovolumic contraction and relaxation phase.

In the presence of regional ischemia, loops in the ischemic segment consistently exhibited an inclination toward the right, while those in the normally perfused area revealed an inclination toward the left. The grade of such an inclination was difficult to quantitate in the present analysis, but the effect of such changes on the shape of the loop can be expressed as the alteration in the area within the loop.

Myocardial asynchrony and deformation of the pressure-length loops. Outward movement during the early phases of systole and with exaggerated shortening in late systole has been noted in the ischemic segment (19–21). The onset of shortening of the normal myocardium occurs earlier because of the unloading effect of the ballooning ischemic myocardium, which acts as an additional elastic slack element from the very beginning of the isovolumic phase of contraction. The ischemic myocardium is unable to support stress during isovolumic systole (22). Similarly, the persistence of shortening in the ischemic segment during relaxation is also associated with outward motion of the normal segment. Thus, the normal and ischemic segments move toward the opposite direction during isovolumic con-

Table 1. Summary of Data

Case	Age (yr) & Sex		Normal Area EDL (mm)	Normal Area ESL (mm)	Normal Area ΔL (mm)	Normal Area W (mm·mm Hg)	Ischemic Area EDL (mm)	Ischemic Area ESL (mm)	Ischemic Area ΔL (mm)	Ischemic Area W (mm·mm Hg)	HR (beats/min)	LVP (mm Hg)	EDP (mm Hg)	EDV (ml)	ESV (ml)	SV (ml)	EF (%)	SW (J)	Coronary Arteriographic Findings (% stenosis)
1	70M	Loc		Diaphragmatic				Anterobasal											
		C	19.4	9.0	10.3	902	23.9	17.4	6.4	722	60	139	12	75	30	45	60	0.67	Left main 90%
		P	22.7	10.6	12.1	743	21.4	19.7	1.7	380	53	135	30	71	32	39	54	0.49	LAD 100%, Coll. R to L
2	67M	Loc		Anterolateral				Inferobasal											
		C	31.9	26.6	5.4	369	25.2	18.0	7.1	808	58	125	15	112	44	68	61	0.61	RCA 99%
		P	31.6	25.1	6.6	266	24.6	20.0	4.6	588	45	100	20	110	47	63	57	0.61	
3	64M	Loc		Inferobasal				Apical											
		C	30.0	26.1	3.9	570	41.0	34.9	6.1	754	46	116	16	125	68	57	45	0.87	LAD 100%
		P	29.7	24.9	4.8	337	41.1	38.9	2.2	282	51	116	20	128	76	52	40	0.84	
4	68F	Loc		Anterobasal				Apical											
		C	32.3	23.1	9.2	809	43.9	37.7	6.2	484	63	124	8	120	68	52	43	0.77	LAD distal 100%
		P	33.7	23.2	10.5	754	40.0	39.7	0.3	35	65	130	19	140	86	54	39	0.62	
5	74M	Loc		Inferobasal				Apical											
		C	29.9	16.6	13.4	999	37.6	27.9	9.7	677	61	143	14	77	41	36	46	0.50	LAD 100%
		P	35.3	20.7	14.7	1041	40.4	40.3	0.2	246	55	138	25	114	75	39	35	0.49	
6	68F	Loc		Diaphragmatic				Anterolateral											
		C	21.7	16.8	5.0	508	29.9	24.0	6.0	770	79	150	15	55	23	32	58	0.50	LAD 100%
		P	22.5	16.9	5.6	448	29.9	27.2	2.7	360	96	145	20	58	30	28	48	0.38	
7	55M	Loc		Inferobasal				Apical											
		C	30.5	26.3	4.2	413	30.6	27.0	3.6	200	82	140	5	122	62	60	49	0.68	LAD 90%
		P	31.6	26.9	4.6	348	31.0	28.7	2.2	129	65	115	27	116	73	43	37	0.49	LCx distal 90%
8	54M	Loc		Diaphragmatic				Anterolateral											
		C	21.0	10.3	10.7	1232	30.1	26.7	3.4	383	61	148	9	103	38	65	63	1.01	LAD 99%
		P	25.2	10.5	14.7	1207	30.2	28.7	1.6	180	55	168	17	112	46	66	58	1.15	
Mean		C	28.6	20.1	8.4	824	32.0	26.1	5.9	600	66	136	11	100	48	52	53	0.70	
± SD			6.3	6.7	3.7	391	6.8	6.6	1.8	205	12	11	4	23	16	12	7	0.17	
		P	30.2	20.3	9.8	751	31.8	29.8	2.0	275	61	131	22	108	59	49	46	0.63	
			5.4	5.9	4.2	434	6.8	7.6	1.2	160	14	18	4	25	20	14	8	0.23	
p			<0.05	NS	<0.005	<0.005	NS	<0.01	<0.002	<0.001	NS	NS	<0.001	NS	<0.05	NS	<0.05	NS	

C = control; Coll. = collateral; EDL = end-diastolic length; EDP = end-diastolic pressure; EDV = end-diastolic volume; EF = ejection fraction; ESL = end-systolic length; ESV = end-systolic volume; F = female; HR = heart rate; ΔL = segment shortening; LAD = left anterior descending coronary artery; LCx = left circumflex coronary artery; Loc = location of selected area; LVP = left ventricular pressure; M = male; NS = not significant; p = statistically significant difference comparing before and after pacing stress; P = post-pacing; RCA = right coronary artery; R to L = right to left; SD = standard deviation; SV = stroke volume; SW = stroke work; W = segmental work index.

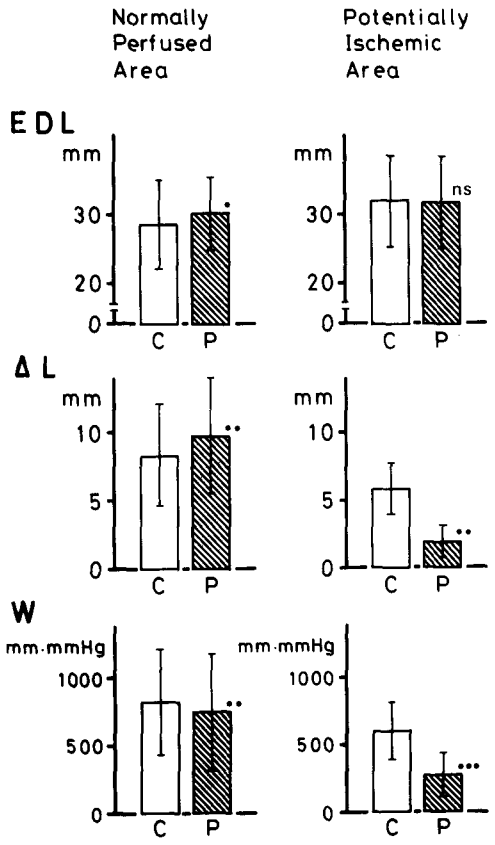

Figure 5. Effects of pacing are shown for the averaged value of end-diastolic segment length (EDL), segmental shortening (ΔL) and segmental work index (W) for representative normal and ischemic sections. In the ischemic section, there was no significant change in end-diastolic segment length in post-pacing beats. Both segmental shortening and work index were markedly reduced, but the reduction in the former was considerably greater than in the latter. In the normal section, end-diastolic segment length was augmented by pacing stress. Despite the significant augmentation of segmental shortening, work index was decreased. Statistical significance; *$p < 0.05$; **$p < 0.01$; ***$p < 0.001$; C = control; P = post-pacing.

traction and relaxation, which may represent a compensatory mechanism to maintain the isovolumic status of the ventricle (21,23).

Tyberg et al. (24) devised an isolated muscle preparation to assess segmental dysfunction in which normal and hypoxic muscles were arranged in series. They demonstrated that the relative motion between the two muscles reflected the dissociation in duration of the active state due to hypoxia. The nonhypoxic muscle contracted earlier against the weaker hypoxic muscle associated with its stretch, whereas the contraction of hypoxic muscle with slow relaxation predominated later with the stretch of the normal muscle. Wiegner et al. (25) further extended this model to a computer-based system and analyzed the mechanics of two muscles in series. They demonstrated that the pattern of shortening during hypoxia becomes polyphasic with a different time course of contraction and relaxation, and muscle length remains constant in late systole only when contractile force and the imposed force are declining at identical rates. In the hypoxic muscle, because of the persisting contractile activity of the muscle and the elastic recoil of passive elements within the muscle, the imposed force decreases less rapidly relative to that of the oxygenated myocardium, resulting in persisting shortening in late systole (25–27).

Segmental work and contractile efficiency. Suga et al. (28–30) considered the two specific areas in the pressure-volume diagram: the area within the pressure-volume loop (external work area) and the area bounded by the end-systolic and end-diastolic pressure-volume lines and the relaxation segment of the pressure-volume loop (area of potential energy) (Fig. 6). The former has been postulated to correspond to the external mechanical work and the latter to the end-systolic elastic potential energy generated during systole and stored at end-systole in the wall of the ventricle. The sum of these two areas, designated as the left ventricular pressure-volume area, is highly related to cardiac oxygen consumption. Suga et al. (28,29) also suggested that potential energy existing in the ventricle at end-systole is convertible into external mechanical work.

According to this concept, late systolic shortening of the ischemic segment indicates that part of the area of potential energy is actually converted into external mechanical work when the ventricle is allowed to eject against an appropri-

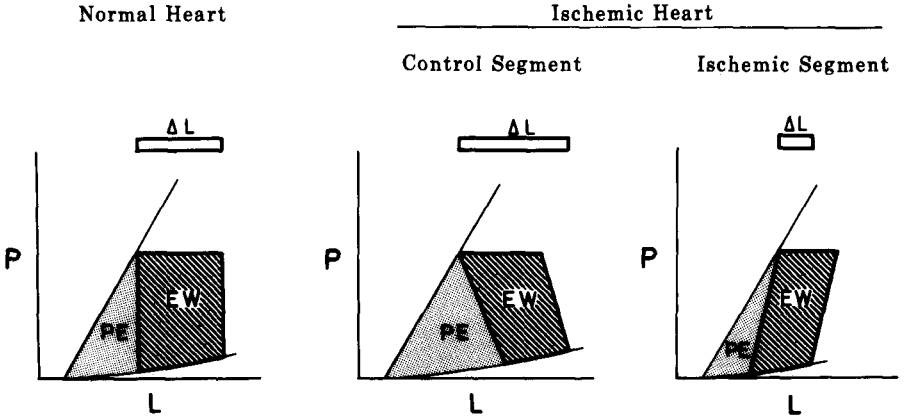

Figure 6. Schematic illustration of external work area (EW) and the area of potential energy (PE) in the normal heart (**left panel**) as conceptualized by Suga et al. (28.29) An inclination of the loop toward the left in the control segment of the ischemic ventricle makes the potential energy larger relative to external work area (**middle panel**), whereas an inclination toward the right in the ischemic segment renders the potential energy smaller relative to the external work area (**right panel**) ΔL = stroke excursion; L = length; P = pressure.

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ately decreasing afterload during relaxation. The work performed by the relaxing ventricle appears to derive from some form of potential energy (30), and therefore, contractile efficiency of the ischemic muscle may be partially improved (Fig. 6).

Mechanism of compensatory augmentation of shortening in normal myocardium. It has been suggested that in the ischemic ventricle, the normally perfused myocardium augments shortening to compensate for the dysfunction in the ischemic myocardium with an early use of the Frank-Starling mechanism (2). Later development of hypertrophy observed in the normal myocardium during healing after myocardial infarction has been attributed to the same mechanism involved in the response to chronic volume overload (31,32).

Our study demonstrates that the normally perfused myocardium in the ischemic ventricle lengthens during relaxation, resulting in an augmentation of the potential energy area relative to the external work area. Thus, the constant enhancement of segmental shortening of the normal area might be attained at the expense of reduced efficiency of contraction (Fig. 6). This response is unlikely to be analogous to the response to simple volume overloading.

Conclusion. Analysis of segmental pressure-length loops generated simultaneously over the entire left ventricular perimeter enabled a detailed analysis of asynchronous wall motion in the ischemic heart. The isovolumic asynchrony of the left ventricle due to the interaction of different myocardial regions with reciprocal temporal relation of contraction and relaxation profoundly modified the shape of regional loops and external segmental work.

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